

Reducing CO₂ Emissions by Managing for Sudden Oak Death...Is It Possible?¹

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Abstract

Forest CO₂ emissions, which have recently become a more regular concern in forest management, can radically increase following pest and disease outbreaks. We inventoried trees in a stand adjacent to an infested area in northern Humboldt County, California, and used a stand-level dynamic disease model to forecast *Phytophthora ramorum*-caused tanoak mortality with and without a proposed treatment to mitigate disease spread and impacts. Using forest growth simulations employing model-predicted mortality rates, a proposed treatment that removes California bay laurel and reduces tanoak stem density results in a substantial increase in the forecasted basal area of tanoak retained after 100 years, along with an increase of up to about 100% in the amount of CO₂ emission equivalents stored as carbon in live trees. While the magnitude of treatment benefit varies depending on how parameters are applied in the disease model and growth simulator, all of the scenarios we attempted resulted in net benefits to retention of larger tanoak and carbon storage.

Introduction

The California Air Resources Board (CARB), under State Assembly Bill 32 (the Global Warming Solutions Act of 2006), is tasked with assuring that California achieves a reduction of greenhouse gas (GHG) emissions to 1990 levels by the year 2020. This equates to reducing emissions to 431 million metric tons CO₂ equivalent (MMTCO₂e) by 1990 and represents a reduction of 15% as compared to a “business as usual” scenario. According to CARB’s 2016 Edition of the GHG Emission Inventory (Released June 2016), California has already reduced its annual emissions from 479.8 in 2006 to 441.5 MMTCO₂e as of 2014. Understanding that forests in California have the capability to store huge amounts of carbon as living biomass (and potentially later as durable wood products), CARB has provided revenues from its Cap and Trade Program to fund forestry projects that forecast net GHG emission reductions. Battles *et al.* (2014) recently estimated that there are 1,050 MMTCO₂e in living trees in California. Thus, even a relatively small proportional change in the amount of carbon stored in forests could have a large positive effect on GHG reduction.

In infested locations where death of mature tanoak (*Notholithocarpus densiflorus* Hook. & Arn.) boles has occurred at high rates over several years due to *Phytophthora ramorum* activity, it is common that dense tanoak re-sprouts perpetually dominate the vegetation (see Cobb, Restoration of Mount Tamalpais Forests Destroyed by the Sudden Oak Death Pathogen, this Proceedings). While the individual tanoak stems from re-sprouting stumps in continually infested forests are most likely to also die from the pathogen before getting beyond sapling stage, more sprouts replace them, and the cycle continues. The continuing presence of *P. ramorum* thus results in substantially less carbon being stored in these forests, where a community of mature tanoak trees does not re-establish.

The dynamics of *P. ramorum* and associated tree mortality in forested stands are largely dependent on the composition and structure of tree communities. With tanoak and California bay laurel (*Umbellularia californica* Hook. & Arn.) both supporting significant amounts of *P. ramorum* pathogen sporangia on

their above-ground tissues, the abundance of these hosts are centrally important in predicting disease effects in forest stands, and stands with lower densities of these hosts generally experience slower disease spread and lower mortality rates (Cobb et al. 2012). With tools to model disease-related mortality of tanoak in forest stands, along with other tools to forecast tree growth and biomass in forests, we can examine how forest management activities can alter disease impacts, and, ultimately, GHG emissions. In this project we examined currently non-infested stands adjacent to a relatively isolated infestation and used these tools to explore the potential effects on disease dynamics and carbon storage under no management and stand treatment scenarios.

Methods

The study area used for the project is a 52-acre unit along a ridge separating Lacks Creek and Stover Creek (both tributaries to Redwood Creek) in northern Humboldt County, California (T8N, R3E, HB&M). To determine initial stand composition, we measured 14 systematically located plots in 2014, using variable radius plots to estimate tree basal area and height and 1/10th-ac fixed plots to estimate density of stems 1 to 4.9 in diameter at breast height (DBH). We classified the unit up into three stand types and used USDA Forest Vegetation Simulator (FVS; v. Feb. 2016; see Crookston et al. 2005) software to summarize initial stand conditions. We then used the average conditions (weighted by stand area) in the unit to input stem density proportions by species into the Sudden Oak Death Dynamics in R (SODDr) model (Ross 2012); this implements the models created by Cobb et al. (2012) in a package for R Statistical Software. We ran the model, starting with a single initial infection location, to predict tanoak mortality over 100 years under the no-treatment scenario and under the scenario in which a treatment is conducted. The treatment consists of cutting down California bay laurel trees in the unit as well as suppressing all sprouts and thinning of tanoak stems, cutting the smallest stems first, to an average stand spacing of 15 by 15 ft. (about 200 stems per acre), with cut trees left on the ground.

Using the tanoak mortality predicted by the model, we adjusted tanoak mortality in FVS at the cycle start/end points. We also selected three different options in FVS for tanoak mortality: 1) mortality occurs uniformly in space and across all size classes of tanoak; 2) mortality occurs in large trees first (supported by Cobb et al. 2012), but is spatially uniform; and 3) mortality occurs uniformly across size classes, but is spatially concentrated at points in the stand. Under a no-treatment scenario, we assumed the disease would first infect the stand in 2017. Under scenarios with treatment, we ran simulations with the disease first infecting the stand in 2017. We also ran simulations in which the disease does not infect the stand until 2032, since the thinned stand may be less likely to become infected.

We adjusted upward tanoak sprouting rates in FVS to reflect actual rates, based on data from an adjacent, similar stand of 45 acres one year after a similar thinning treatment. Since FVS simulates sprouting of tanoak after thinning, but not after mortality, we added rates of natural regeneration corresponding to the sprouting rate per individual and to the number of individuals predicted to die in each cycle in an initial simulation. We generated carbon reports with the Fire and Fuels extension (FFE) in FVS, using the option of Jenkins et al. 2003 biomass equations, and calculated CO₂e for each of live biomass (99.7-99.9% of which is estimated to be in trees), dead material, and their sum.

Results

In 2014, there was an average of 558 stems per acre of tanoak at least 1 in diameter at breast height (4.5 ft; DBH). The thinning treatment scenario removes ~390 tanoak stems per acre at least 1 in DBH, plus all smaller tanoak stems; this produces a stand with tanoak in the 9-40 in DBH range, plus components of Douglas-fir (*Pseudotsuga menziesii* var. *menziesii* (Mirb.) Franco), Pacific madrone (*Arbutus menziesii*

Pursh), and giant chinquapin (*Chrysolepis chrysophylla* Dougl. ex Hook.). California bay laurel trees comprised about 2.5% of the initial stems.

The disease model predicts that tree mortality occurs at a faster rate under untreated stand conditions (fig. 1a), but the proportion of the stand stems that are tanoak is similar after 100 years whether or not the treatment is done (also see table 1). However, the thinning treatment results in a stand structure with fewer, larger tanoak trees; quadratic mean diameter under the thinning scenarios is projected to be consistently higher (data not shown). Similarly, the forecasted basal area of tanoak in the stand in 2117 is substantially higher in thinned stands than untreated ones—from 35% to 131% higher with thinning undertaken in 2017 than without (table 1). The thinning treatment is also consistently projected to have a carbon benefit. After 100 years of SOD dynamics and forest growth, the thinned stand is projected to have a maximum benefit (out of the simulated scenarios) of 23,624 metric tons of the CO₂ equivalent in live trees (fig. 1b), and a minimum benefit of 6,917 metric tons (scenario 3 in table 1). Live-tree carbon differences between disease introduction times under the thinning scenario are negligible (fig. 1b).

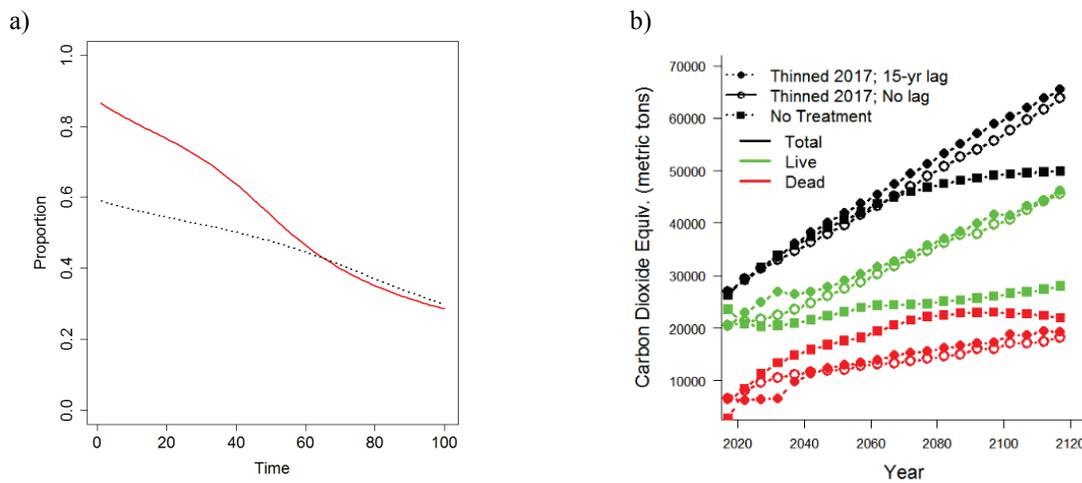


Figure 1—a) Predicted proportion of total tree stems that are tanoak in untreated (red line) and thinning treatment (black dotted line) conditions over time (in years); b) Forecasts of CO₂ equivalent present in the stand, 2017 through 2117, according to scenario 3 described in table 1.

Table 1—Stem density and basal area of tanoak forecast in 2117 under different scenarios

Scenario	Tanoak projected in year 2117	
	Stem density (trees per acre)	Basal area (square ft ac ⁻¹)
Mortality uniform in space and among size classes (1)		
Stand is thinned; disease arrives in 2017	189	397
No treatment; disease arrives in 2017	189	265
Mortality uniform in space; large trees killed first (2)		
Stand is thinned; disease arrives in 2017	153	343
Stand is thinned; disease arrives in 2032	161	359
No treatment; disease arrives in 2017	182	147
Mortality clustered; uniform among size classes (3)		
Stand is thinned; disease arrives in 2032	246	394
No treatment; disease arrives in 2017	192	293

Discussion

Our results suggest that removal of California bay laurel and thinning of tanoak in advance of *P. ramorum* arrival into a stand can have positive effects on retention of tanoak in larger size classes, and these effects also come with a benefit in terms of the amount of carbon retained in the stand. In this project, GHG reduction benefits were forecast under several different scenarios that account for some of the potential variability in mortality patterns. For future studies, it would be useful to include within-stand spatial heterogeneity of host distribution in the disease mortality model. Current work is underway at Humboldt State University to make the SODDr package more conducive to this. Additional attention should also be given to tanoak sprouting dynamics in FVS and regional differences. The potential avoidance of CO₂ emissions associated with similar treatments is potentially far higher than estimated here because tanoak mortality from *P. ramorum* also increases the likelihood that wildfires are less manageable and their effects more severe (Valachovic et al. 2011). Thus, management targeted toward areas vulnerable to *P. ramorum* can achieve several goals beyond reducing the spread of the disease through the landscape.

Acknowledgments

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